

Rb<sub>1</sub>. Physiological saline containing ginsenoside Rb<sub>1</sub> was filled in advance in the said osmotic minipump, and ginsenoside Rb<sub>1</sub> in a dose of 60  $\mu$ g/day or 6  $\mu$ g/day was continuously infused into the blood stream through the catheter placed in the left femoral vein for 28 days. Flow rate of the ginsenoside Rb<sub>1</sub>-containing solution was 0.25  $\mu$ l/hour. Control animals with MCA permanent occlusion (ischemic control animals) and sham-operated animals received the same amount of physiological saline.

#### Example 2 (Water maze tests)

After the MCA permanent occlusion according to the method of the inventors of the present invention (Sakanaka and Tanaka) (Igase, K. et al., J. Cereb. Blood Flow Metab., 19, 298-306, 1999; Zhang B. et al., J. Stroke Cerebrovasc. Dis., 7, 1-9, 1998), water maze tests were performed for 4 days at the 2nd week and the 4th week, respectively, and place navigation abilities of the SH-SP rats were determined.

Results are shown in Fig. 1. In Fig. 1, the left drawing shows the results of the 2nd week and the right drawing shows the results of the 4th week after permanent MCA occlusion. In Fig. 1, closed circles (●) indicate the results of rats with sham-operation; and open circles (○) indicate the results of MCA-occluded rats administered with only physiological saline; closed squares (■) indicate the results of MCA-occluded rats

administered with ginsenoside Rb<sub>1</sub> in a dose of 6  $\mu$ g/day and open squares (  $\square$  ) indicate the results of MCA-occluded rats administered with ginsenoside Rb<sub>1</sub> in a dose of 60  $\mu$ g/day. Data are represented as a mean  $\pm$  SE. Statistical analyses were conducted by ANOVA + Fisher's PLSD.

No significant differences in swimming speed were detected among the four experimental groups.

#### Example 3 (Measurement of ratio of lesion)

After termination of the water maze tests at the 4th week, the SH-SP rats were anesthetized with chloral hydrate, and they were perfused and fixed transcardially with 0.1 mole phosphate buffer containing 4% paraformaldehyde. The brains were dissected out and cerebrocortical infarcted areas were photographed. Areas of the left cerebral hemispheres and the left cerebrocortical infarct lesions were measured on the photographs by using an image analysis device. The left cerebrocortical infarcted areas were divided by the left cerebral hemispheric areas to calculate ratios of the cerebrocortical infarction (%). Results are shown in Fig. 2. Data are represented as mean  $\pm$  SE. Statistical analyses were performed by Mann-Whitney U-test.

An actual case of a cerebral infarct lesion of the group administered with physiological saline and an actual case of a cerebral infarct lesion of the ginsenoside Rb<sub>1</sub> (6  $\mu$ g/day)-

administered group are shown in Fig. 3A and Fig. 3B, respectively.

Fig. 4 is a schematic drawing summarizing the results of the present experiments. In rats administered with physiological saline, the size of cerebral infarction remained large, and in the water maze tests, it took a long time for the rats to escape onto the goal platform. Contrary, in rats administered with ginsenoside  $Rb_1$  of the present invention, the infarct area was reduced, and as a result, in the water maze tests, only short time was required for the rats to arrive at the goal platform.

The isolated brain was embedded in paraffin, and paraffin sections,  $5\mu\text{m}$  thick, at the level approximately  $2.8\text{ mm}$  posterior to bregma including the cerebrocortical infarct lesion were prepared. Then, we measured the cerebrovascular area per  $1.27\text{ mm}^2$  of the ischemic penumbra rescued by intravenous administration of ginsenoside  $Rb_1$ . This was done for each of the cerebral hemispheres of the groups to which ginsenoside  $Rb_1$  had been administered intravenously (i.e. the brain tissues had been rescued by administering ginsenoside  $Rb_1$ ) (Fig. 5). Four sheets of differential interference contrast micrograph were used for measurement (Fig. 6), and the ratio of the cerebrovascular area was calculated (Table 1). The ratio of cerebrovascular area was also measured in the control hemisphere (intact side). As indicated in Table 1, the ratio of cerebrovascular area showed